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Patterns of spread of coral disease in the Florida Keys

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Abstract

Reefs in the Florida Keys are experiencing a dramatic increase in the number of localities and number of species with coral disease. In extensive surveys from Key Largo to Key West in 160 stations at 40 randomly chosen sites, there has been a dramatic increase in (1) the number of locations exhibiting disease (82% of all stations are now affected, a 404% increase over 1996 values), (2) the number of species affected (85% of all species are now affected, a 218% increase over 1996 values), and (3) the rate of coral mortality (the deep fore-reef at Carysfort experienced a 60% reduction of living coral cover during the survey). Two null hypotheses (1) that the incidence of disease has remained constant through time and (2) that the apparent increase in disease is due to a lack of comparable earlier data, are both falsified. Different diseases exhibit different patterns of spread: some diseases (e.g. black band) exhibit low incidence and jump rapidly between sites; other diseases (e.g. white pox) exhibit patchy distributions and increase in frequency at affected sites from one year to the next. The central question of why so many corals are becoming simultaneously susceptible to a host of marine pathogens remains unanswered.

Introduction

Although disease is a part of the natural environment, there is a gathering impression among both terrestrial ecologists (McCallum & Dobson, 1995) and marine scientists (Epstein, 1998; Hayes & Goreau, 1998; Harvell et al., 1999) that disease is playing an increasingly important role in regulating the population size and demographic characteristics of wildlife populations world-wide. Examples of species reductions are occurring in both terrestrial environments, such as the loss of many species of amphibians in Central America due to a chytrid fungal disease (Berger et al., 1998), and in the marine environment such as disease-induced mass mortalities recorded in the Caribbean among sea urchins (Lessios et al., 1984), sea fans (Nagelkerken et al., 1997a,b; Kim & Harvell, in press), and sea grasses (Roblee et al., 1991).

Epizootics have also been reported affecting reef-building corals (Richardson, 1998; Goreau et al., 1998; Harvell et al., 1999). Extremely heavy losses of the two commonest species of the Caribbean acroporid corals, *Acropora palmata* and *A. cervicornis*, have been documented in St. Croix (Gladfelter, 1982), Belize (McClanahan & Muthiga, 1998), and Jamaica (Hughes, 1994; Greenstein et al., 1998) as a result of white band disease. Severe reductions in *A. palmata* are occurring in Florida due to a new disease, white pox (Holden, 1996; Porter et al., 2002). *Montastraea faveolata* populations are diminishing in part due to yellow blotch disease (Santavy et al., 1999), and heavy losses have been inflicted on several coral species due to white plague (Richardson et al., 1998a,b).

The rapid loss of corals in some locations, such as on Jamaica coral reefs (Hughes, 1994) has been accompanied by ecological phase shifts from coral

dominated substrates to algal dominated substrates. Causes for the increase in algae have been ascribed to the loss of herbivores (both grazing fish and urchins), that is by 'top-down' controls (Hughes et al., 1999; Aronson & Precht, 2000) *versus* an increase in available nutrients (both phosphorous and nitrogen) that is by 'bottom-up' controls (Lapointe, 1997, 1999). While it is true that either grazer loss or nutrient increase results in greater algal biomass, neither of these mechanisms addresses the role of coral disease in creating substrate for algal colonization.

Surveys which attempt to detect the way in which an ecosystem changes over time require the collection of base line data in an unbiased fashion. Without 'before' data with which to compare 'after' conditions, change detection is impossible. Furthermore, an unbiased sampling design allows one to quantify both the direction and magnitude of change. Such information can be especially useful in management or regulatory decision making processes. In this paper, we will describe a field method employed to map the distribution of coral diseases throughout the Florida Keys coral reef ecosystem, and to quantify their change through time.

Materials and methods

Site selection and station installation

The U.S. Environmental Protection Agency Coral Reef Monitoring Program (CRMP) was designed to assess the status and trends of Floridian coral reefs. Three principles govern site selection in studies designed to detect change (Warwick & Clarke, 1991; Clarke, 1993): (1) site selection must include localities scattered throughout the region of interest, (2) site selection must be conducted in an unbiased fashion, and (3) site selection must include a sufficient number of samples to detect subtle change. The region of interest is large, including coral reef habitats distributed throughout the 350 km length of the Florida Keys, an area greater than 9600 km^2 (Fig. 1). To satisfy these criteria, a stratified random (EMAP) site selection regimen (Overton et al., 1991; Porter et al., 2002) was used to choose the coordinates of 40 reef sites distributed throughout five EPA Water Quality Segments (Hankinson & Conklin, 1996) within the Florida Keys National Marine Sanctuary (Fig. 1). A vast majority of Floridian reefs may be classified as belonging to one of four reef types: (a) offshore shallow reefs; (b) offshore deep reefs, (c) patch reefs, and (d) near-shore hard-bottom habitats (Porter & Tougas, 2001). The 40 randomly selected sites were sufficient to included all of the reef types found naturally in each of the five Water Quality Segments (Fig. 1).

At each randomly selected site, four sampling stations were established. From the latitude and longitude of the selected locality, a random compass rose heading was chosen, and a snorkeler swam in this direction until encountering the first reef. This location then became the first station. Three additional stations were chosen at this same location and depth. Practical considerations resulted in the exact locations of the remaining three stations at the site being chosen by haphazard means within the general vicinity of the first randomly chosen station. At each station, a pair of stainless steel survey pins were implanted in the bed rock 20–22 m apart, perpendicular to the depth contour or coast line. This selection process resulted in the establishment of 160 stations within 40 reef sites in the Florida Keys National Marine Sanctuary.

Static and dynamic trends in the geographic distribution of coral disease

Stations are sampled annually (Porter et al., 2002). During sampling, a 2-m long pole is inserted onto the reference stakes at either end of the station, with the reference stake located at the center of the pole. Three lines are stretched between the two poles, one down the left and right ends from pole to pole and one down the middle between the survey pins. When erected, the survey grid looks like a clothes line with three parallel lines 1 m apart stretched between the two poles 20 m apart. Two kinds of surveys are conducted within this sampling grid. Inside of the 40 m^2 area defined by the two poles (2 m width$\times$20 m length), inventories are conducted on (a) the presence or absence of all scleractinian and milleporine coral species, and (2) the presence or absence of coral disease on any colonies of any of these species. These observations are made by two qualified observers and recorded on underwater writing paper. To assure intercomparability between stations and years, surveys were standardized in the following manner: each observer spent 15 min swimming the transect recording coral species and coral disease (approximately 7.5 min down the station on one side, and 7.5 min back on the other). This observation period was followed by a 5-min confirmation period during which taxonomic questions and observational discrepancies were resolved. For each

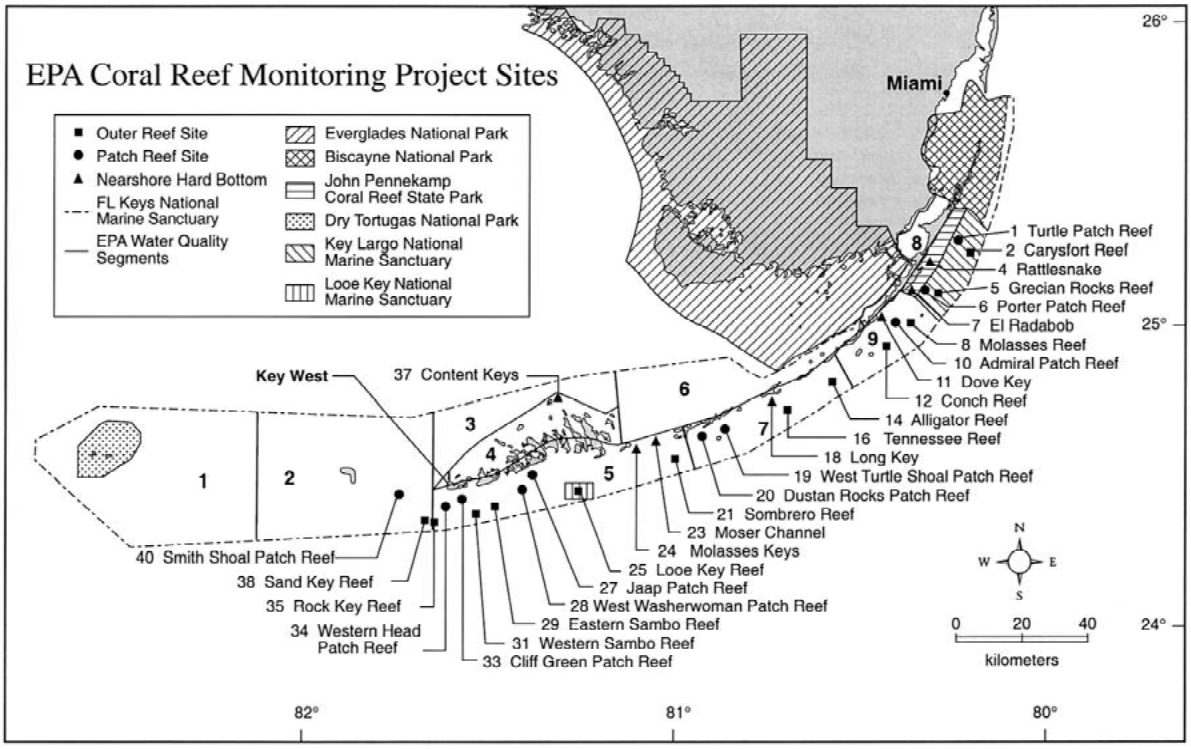

Figure 1. Forty randomly chosen sites within the Florida Keys National Marine Sanctuary were surveyed annually for coral biodiversity, live coral cover, and the presence of three types of coral disease (Table 1). Reef sites (solid squares) include both an offshore shallow (2–7 m) site and an offshore deep (10–20 m) site.

station, data were reported as the union of the two lists, that is, the concatenation of both observer's lists. During the 1996 field season, based on a shortage of trained personnel, of the 160 stations in the complete survey, 132 stations were double counted and the remaining 28 were counted by a single observer. In subsequent years, all 160 stations were double counted. To make the surveys comparable from site to site, the 15-min swim-survey protocol was adhered to with strict uniformity.

Disease definition and disease types

In the CRMP survey, only clear and unequivocal signs of disease were recorded. Coral disease was also carefully distinguished from coral bleaching (Brown, 1997), which superficially can look like disease. To make a disease determination, observers looked for active tissue necrosis. Often this was accompanied by bared skeleton, mucus production and partial disintegration of polyps. Blemishes, slight discolorations and small, cryptic examples of disease were not scored. We chose characteristics that were as pathognomic as possible for underwater determinations. Anchor scrapes, parrot fish bites, predatory snail wounds and old scars of unknown origin were not scored as disease.

Due to time constrains underwater, and also due to the poorly defined nature of many coral diseases (Richardson, 1998), diseases were assigned to one of three clearly identifiable categories: black band disease, white diseases and other diseases (Table 1). Black band disease is caused by a cyanobacterial (*Phormidium corallyticum*) dominated microbial mat (Table 1), whereas the remaining two categories, white diseases and other diseases, are more than likely caused by several different pathogens (Table 1). Many of the white diseases are virtually impossible to differentiate in the field. For instance White Band Types I and II, and White Plague Types I and II, require information on the rate of spread, and therefore are impossible to distinguish in the field. Often coral tissue loss and damage can not be assigned to either white disease or black band disease. These injuries include a variety of lesions that may be associated with (as yet uncharacterized) newly emerging coral diseases. The

Table 1. Coral disease conditions scored during the Coral Reef Monitoring Project Station Species Inventories

CRMP Disease Category	Common Name	Pathogen	Reference
Black line disease	Black Band	Microbial consortium	Richardson (1998)
White line diseases	White Pox	Unknown	Holden (1966)
	White Band, Type I	Unknown	Gladfelter (1982)
	White Band, Type II	Gram negative bacteria, Including *Vibrio charcharia*	Ritchie & Smith (1998)
	White Plague, Type I	Mix of gram negative bacteria	Dustan (1977)
	White Plague, Type II	*Sphingomonas* sp. nov.	Richardson et al. (1998a)
Other diseases	Yellow Blotch	Unknown	Santavy et al. (1999)
	Dark Spot	Unknown	Goreau et al. (1998)
	Ridge Mortality	Unknown	Goreau et al. (1998)
	Red Band	*Oscillatoria*?	Goreau et al. (1998)
	Rapid wasting	Fungal/Predation	Cervino et al. in press
	Neoplasia	Cancer?	Goreau et al. (1998)

Criteria for disease designation: Active tissue mortality, tissue necrosis, bared skeleton, mucus production, bisected or partial polyps.

terminology used to describe these new diseases is still in flux. These diseases have been given descriptive names such as 'ridge mortality', 'red stripe', 'coenosarc swelling' and 'dark spot'. For purposes of our underwater survey, we lumped these as Other Disease. Splitting of these CRMP disease categories required a prohibitive increase in survey time, and, further, resulted in a substantial number of misidentifications. Data were recorded as the presence of the three types of disease for each species, station and year. Since each site consists of four stations, these data were expressed as proportionality of affected stations within a site from 0.0 to 1.0.

Quality assurance/quality control

Two quality assurance exercises were conducted. (1) A visual test using slides was administered to each coral disease counter. The test required correct identification of both the coral species and disease condition shown on the projection screen. Healthy as well as diseased corals were included in the slide set to assess the frequency of false disease reports. (2) Coral disease counters were also tested in the field. On the Sand Key Shallow coral reef, the four counters (A, B, C and D) counted all four stations two times. After the first count of four stations, the underwater survey lines were taken up, and then reset by a different set-up team. While the survey lines were being reset, the observers were assigned new counting partners at random and the site was sampled a second time. This exercise resulted in station counts by four different buddy pairs (A/C, B/D, A/D and B/C).

Live coral cover

On each station, a weighted transect chain was laid under each of the three transect lines. A video camera was then swum directly over the transect chain while being held a constant height of 40 cm above the reef. The camera pointed directly down at the substrate and followed the contours of the reef as it was moved at a constant speed between the two poles at either end of the station (Porter et al., 2002). From the resulting video footage, approximately 120 frames were grabbed from the video tape and stored on CD ROM for counting. From this library of images, abutting or non-overlapping images were selected for analysis. Ten random points were generated and applied to each frame, and the identity of the substrate category beneath the point was scored. In addition to the species level taxonomy of the corals, several additional biotic categories were also enumerated: (1) macroalgae, (2) hydrocorals, (3) octocorals, (4) sponges, (5) zooanthids, and (6) substrate. Percent cover for each substrate category was determined as the average of its proportional representation on each of the three parallel transect lines.

Some of the more easily recognizable disease conditions can also be identified from the video record (see the video images in Figure 2 for an example of white plague, and Figure 3 for an example of white

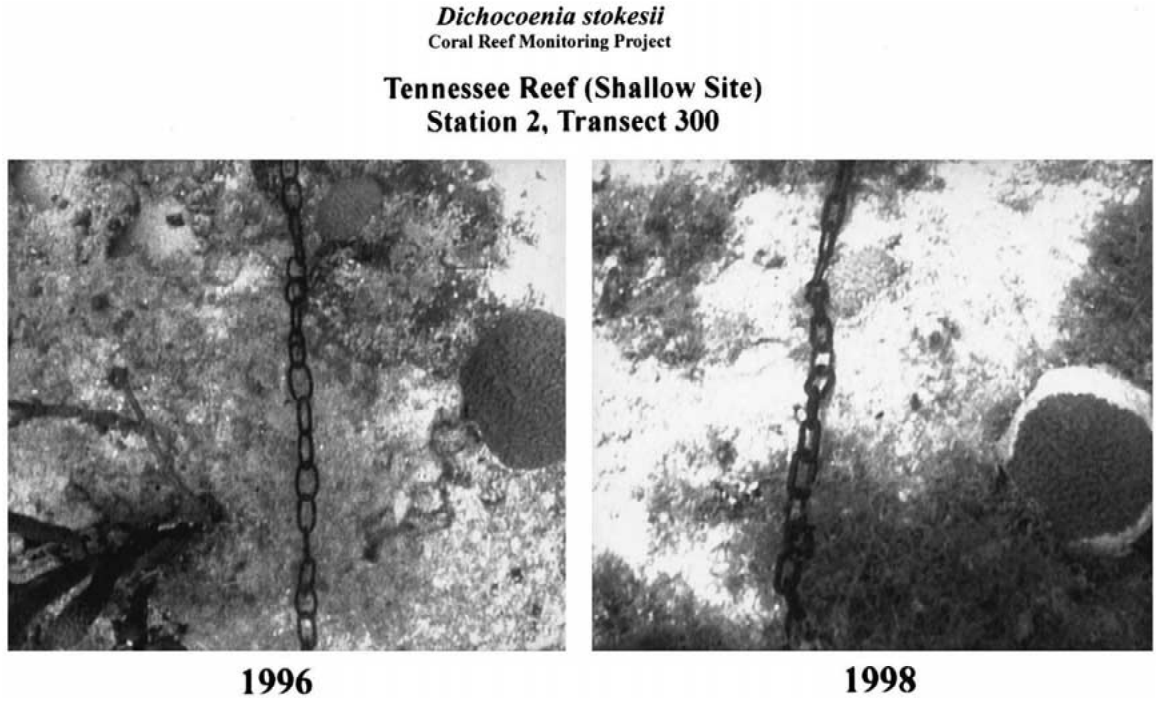

Figure 2. During our survey, *Dichocoenia stokesi* colonies suffered high partial and whole mortality from white plague, as seen in these paired images from Station 2 at the Tennessee Reef Shallow Coral Reef Monitoring Site, 1996–1998.

pox). To test the utility of using video to assess the presence of specific coral diseases, videos from shallow water offshore sites were scanned visually, and the presence of white pox lesions noted for colonies of *Acropora palmata*.

Results

Quality assurance/quality control

The field exercise produced high agreement among coral disease observers. Of the 24 species recorded on the shallow Sand Key Reef site, 13 species (slightly more than 50%) had diseased individuals. All four observers identified diseased individuals for 10 of these 13 species; three observers identified diseased individuals in the eleventh species; two observers identified diseased individuals in the twelfth species; and one observer found a diseased specimen in the thirteenth species. Since all data for the disease survey are reported as the union of the two observer's lists, Table 2 presents a comparison between results from individual counters as well as between paired observers. The data show a very high concordance between individual counters for both the number of species and the number of stations with each type of disease (Table 2). This result was expected based on the narrow range of high test scores (94.2%±4.8 St.Dev.) achieved by all of the counters on the visual test. As one would expect, counts made by two qualified observers were slightly higher than counts made by one, but in no instance were single-observer counts significantly different than double-observer counts (Table 2). On average, the effect of single-observer counting was to reduce the result by one standard deviation of the mean (Table 2).

Static and dynamic trends in the geographic distribution of coral disease

Between 1996 and 1998, the incidence of coral disease in the Florida Keys increased dramatically (Table 3). Whereas in 1996, among the 160 stations (at 40 sites) only 26 stations contained diseased individuals (16% of the stations), in 1997 this number had risen to 95 (59% of the stations), an increase of 265% over the 1996 figures. By 1998, this number had risen again to 131 stations (82% of the survey), a 404% increase over and above the 1996 values. The trend is the same regardless of whether the incidence of disease is computed on a per station basis (Table 3) or on a per site

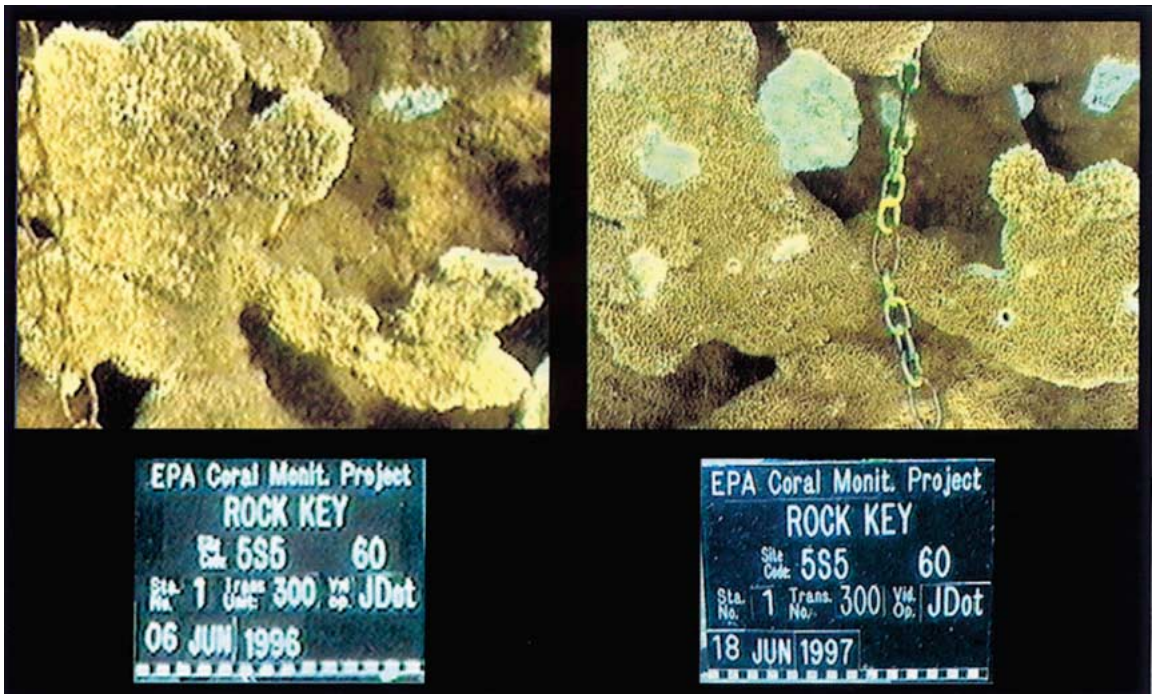

Figure 3. The emergence of white pox on *Acropora palmata* colonies at Rock Key Reef off Key West, FL can be seen in these paired images, grabbed from the CRMP transect video taken in 1996 (left) and 1997 (right).

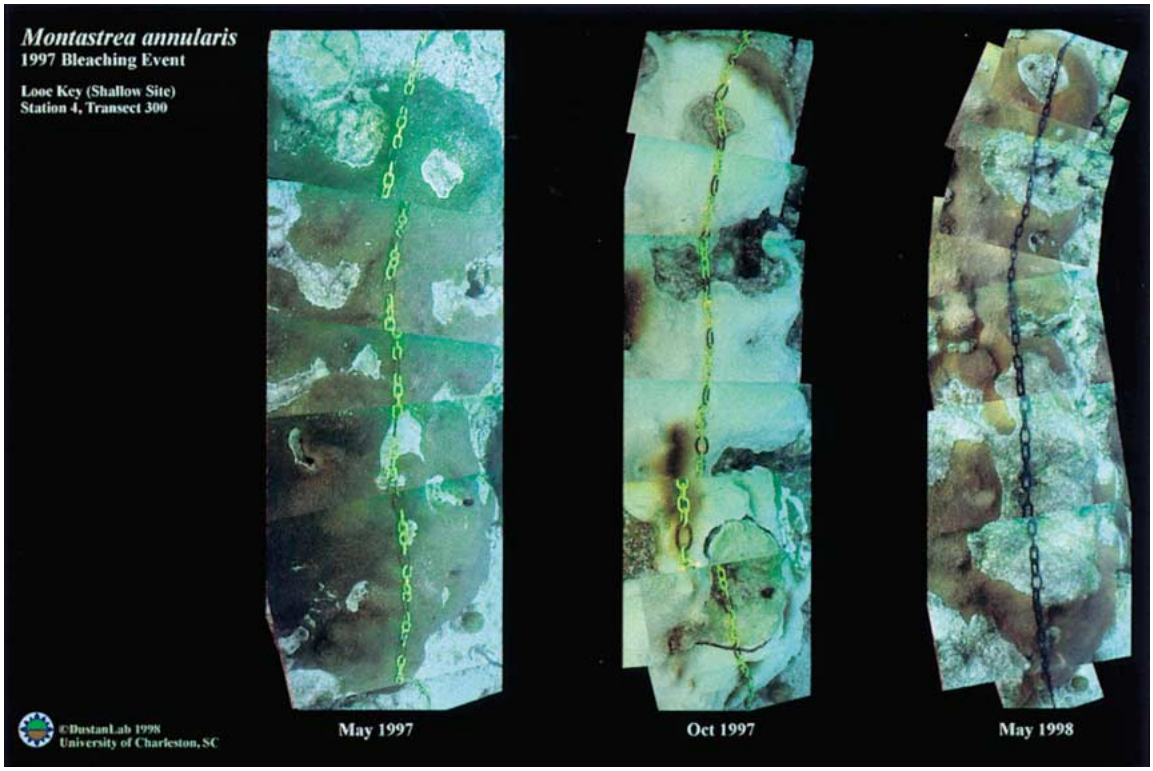

Figure 10. The potential interaction between coral bleaching and disease can be seen in this montage of images from 1997 to 1998. Healthy colonies of *Montastraea annularis* [left (May, 1997)] bleached due to elevated late summer sea surface temperatures [middle (October, 1997)]. This colony also contracted black band disease (middle, lower part of the image). By May, 1998 (right), most of the colony had recovered, but the black-band damaged tissue did not.

Table 2. Quality assurance field tests on both the number of coral species and the number of survey stations with coral disease. Each of the four counter's individual results and their paired results are listed for the presence of white diseases (WH), black band disease (BB), other diseases (OD), and total diseases (TD)

Disease category					Mean±SD
Number of Coral Species with Disease					
Individual Counter	A	B	C	D	
WH	7	7	7	7	7.00±0.00
BB	0	0	0	0	0.00±0.00
OD	4	2	3	4	3.25±0.96
TD	11	9	10	11	10.25±0.96
Counter Pair	A/C	B/D	A/D	B/C	
WH	7	7	7	7	7.00±0.00
BB	0	0	0	0	0.00±0.00
OD	4	4	5	6	4.75±0.96
TD	11	11	12	13	11.75±0.96
Number of Stations with Disease					
Individual Counter	A	B	C	D	
WH	3	3	3	3	3.00±0.00
BB	0	0	0	0	0.00±0.00
OD	3	1	2	2	2.00±0.82
TD	4	3	3	3	3.25±0.50
Counter Pair	A/C	B/D	A/D	B/C	
WH	3	3	3	3	3.00±0.00
BB	0	0	0	0	0.00±0.00
OD	3	2	4	3	3.00±0.82
TD	4	3	4	4	3.75±0.50

Table 3. The number of EPA Coral Reef Monitoring Stations in the Florida Keys with coral disease (WH, white diseases; BB, black band disease; OD, other diseases; and TD, total diseases). The percent increase, 1996–1998, is listed at the bottom; the percent of affected stations is listed in parentheses

Year	Disease condition			
	WH	BB	OD	TD
Number of Stations in the Florida Keys with Disease				
1996	**7** (4%)	**7** (4%)	**16** (10%)	**26** (16%)
1997	**61** (38%)	**12** (8%)	**65** (41%)	**95** (59%)
1998	**97** (61%)	**28** (18%)	**92** (58%)	**131** (82%)
Increase (1996–1997)	771%	71%	306%	265%
Increase (1997–1998)	59%	133%	42%	38%
Increase (1996–1998)	1286%	300%	475%	404%

basis (Table 4). Whereas in 1996, only 16 sites (40% of the sites) had diseased colonies (i.e. TD $\geq$0.025), by 1998, almost all of the sites (37 or 93%) were affected by disease, an increase of 131% (Table 4).

The three individual disease categories all follow the same trend as the total disease category. Between 1996 and 1998, white diseases increased the most, rising from 7 to 97 stations, but both black band (increasing from 7 to 28 stations) and other diseases (increasing from 16 to 92 stations) also expanded their distribution substantially (Table 3).

Although most of the increase occurred between 1996 and 1997, large increases also occurred between 1997 and 1998. Expressing these data on a proportional basis (Table 4), white diseases jumped from a mean proportional representation in the 40 survey sites from 4.4% in 1997 to 60.6% in 1998. Black band exhibited more modest increases in proportional representation from 4.4 to 17.5%. Other diseases rose from 10.0 to 57.5% (Table 4).

Between 1996 and 1998, the coral disease increases measured by in our survey are quite large. Not surprisingly therefore, statistical analysis reveals highly significant increases in all disease categories (Table 5). These rising trends (Fig. 4), clearly and unequivocally falsify the null hypothesis that there has been no significant increase in coral disease in the Florida Keys.

Data were not taken on the percent of colonies affected by disease. However, our careful observations within the stations led to the distinct impression that in 1996 most of the incidences of disease were exhibited by only one individual of a species within a station, whereas in 1998 most of the incidences of disease were manifested by many colonies of each species.

Table 4. Proportion of EPA Coral Reef Monitoring Stations with coral disease at each CRMP Site (Fig. 1), 1996–1998. Proportion is calculated as the fraction of the four stations at each site (0.00, 0.25, 0.50, 0.75 or 1.00) with this disease condition (WH, white disease; BB, black band disease; OD, other diseases; and TD, total diseases)

Site name	Lat.	Long.	1996				1997				1998			
			WH	BB	OD	TD	WH	BB	OD	TD	WH	BB	oD	TD
1. Turtle Patch Reef	25° 17.6647′	80° 13.1481′	0	0	0	0	0.50	0	0.75	1	1	0	0.50	1
2. Carysfort Reef, D.	25° 13.2481′	80° 12.5915′	0	0	0	0	0.75	0	0.75	1	1	0	0.50	1
3. Carysfort Reef, S.	25° 13.3339′	80° 12.5851′	0.25	0	0	0.25	0.75	0	0.50	0.75	0.75	0	0.75	1
4. Rattlesnake	25° 10.4150′	80° 20.8500′	0	0	0	0	0	0	0	0	0	0	0	0
5. Grecian Rocks, S.	25° 06.4528′	80° 41.4155′	0.25	0.25	0	0.50	0.75	0	0.25	0.75	0.75	0.25	0.50	1
6. Porter Patch	25° 06.1899′	80° 19.4586′	0	0	0	0	1	0	1	1	1	0	0.75	1
7. El Radabob	25° 07.2068′	80° 22.6937′	0	0	0	0	0	0	0	0	0	0	0	0
8. Molasses Reef, D.	25° 00.4311′	80° 22.5338′	0	0	0	0	0	0.50	0.75	0.75	0.75	0	0.25	0.75
9. Molasses Reef, S.	25° 00.5250′	80° 22.5890′	0.25	0	0	0.25	1	0.25	0.75	1	1	0	1	1
10. Admiral Pacth	25° 02.6480′	80° 23.6850′	0	0.50	0	0.50	0	0	0.50	0.50	0.25	0	0.75	0.75
11. Dove Key	25° 02.6793′	80° 28.1025′	0	0	0	0	0	0	0.25	0.25	0.75	0	0.25	0.75
12. Conch Reef, D.	24° 57.1114′	80° 27.0807′	0	0	0	0	0.50	0.50	0.25	0.75	0.75	0.50	0.50	1
13. Conch Reef, S.	24° 57.3150′	80° 27.4810′	0	0	0	0	0.25	0	0	0.25	0.50	0	0	0.50
14. Alligator Reef, D.	24° 50.7100′	80° 37.2563′	0.25	0	0.25	0.50	0	0	0.50	0.50	0.50	0.25	0.75	1
15. Alligator Reef, S.	24° 50.7723′	80° 37.3812′	0	0	1	1	0	0	0.25	0.25	0.50	0	0	0.50
16. Tennessee Reef, D.	24° 45.1621′	80° 45.4696′	0	0.25	0	0.25	0	0	0.25	0.25	1	0.50	0	1
17. Tennessee Reef, S.	24° 44.6980′	80° 46.8730′	0	0	0	0	0	0	0.25	0.25	1	0	0.25	1
18. Long Key	24° 47.8340′	80° 47.0400′	0	0	0	0	0	0	0	0	0.50	0	0.50	0.50
19. West Turtle Shoal	24° 41.9572′	80° 58.0127′	0	0	0.25	0.25	0	0	0	0	0.25	0.50	1	1
20. Dustan Rocks	24° 41.3676′	81° 01.8101′	0	0	0.50	0.50	0	0	0.25	0.25	1	1	1	1
21. Sombrero Reef, D.	24° 37.3347′	81° 06.7040′	0	0	0	0	0	0.25	0.25	0.50	0.50	0.25	1	1
22. Sombrero Reef, S.	24° 37.5310′	81° 06.6240′	0.25	0	0	0.25	0	0	0.75	0.75	0.50	0.75	0.75	1
23. Moser Channel	24° 41.3470′	81° 10.0546′	0	0	0	0	0	0	0	0	0	0	0.50	0.50
24. Molasses Keys	24° 40.5371′	81° 11.4294′	0	0	0	0	0	0	0	0	0.25	0	0	0.25
25. Looe Key Reef, D.	24° 32.5230′	81° 24.9178′	0.25	0	0.25	0.25	1	0.25	0.50	1	0.75	0	0.25	0.75
26. Looe Key Reef, S.	24° 32.7157′	81° 24.4766′	0.25	0.50	1	1	0	0	0.50	0.50	0.75	0.75	1	1
27. Jaap Patch Reef	24° 35.1421′	81° 34.9568′	0	0	0	0	0	0	0.50	0.50	0	0.75	1	1
28. W. Washerwoman	24° 32.8480′	81° 35.1934′	0	0	0.25	0.25	0	0	0.50	0.50	0.25	1	1	1
29. Eastern Sambo, D.	24° 29.3029′	81° 39.9514′	0	0	0	0	1	0.25	0	1	0.25	0.50	0.50	0.75
30. Eastern Sambo, S.	24° 29.5013′	81° 39.8139′	0	0	0	0	0.50	0	0.25	0.75	0	0	0	0
31. Western Sambo, D.	24° 28.6808′	81° 43.0275′	0	0	0.25	0.25	1	0.25	0.75	1	1	0	0.75	1
32. Western Sambo, S.	24° 28.7708′	81° 43.0293′	0	0	0	0	1	0	0.75	1	1	0	0.75	1
33. Cliff Green Patch	24° 30.2160′	81° 46.0590′	0	0	0	0	0.25	0	1	1	0.50	0	0.75	0.75
34. Western Head	24° 29.8625′	81° 48.3343′	0	0	0	0	1	0	0.75	1	1	0	1	1
35. Rock Key Reef, D.	24° 27.1929′	81° 51.4076′	0	0	0	0	0	0.25	0.50	0.50	0.50	0	1	1
36. Rock Key Reef, S.	24° 27.2852′	81° 51.5890′	0	0	0	0	1	0	0	1	1	0	0	1
37. Content Keys	24° 49.3230′	81° 29.3350′	0	0	0.25	0.25	0	0	0.25	0.25	0	0	1	1
38. Sand Key Reef, D.	24° 27.1005′	81° 52.7909′	0	0	0	0	1	0.25	0.50	1	0.75	0	1	1
39. Sand Key Reef, S.	24° 27.1190′	81° 52.6500′	0	0	0	0	1	0	1	1	1	0	0.75	1
40. Smith Shoal	24° 43.1895′	81° 55.1757′	0	0.25	0	0.25	1	0	0.50	1	1	0	0.75	1
		Mean:	**0.044**	**0.044**	**0.100**	**0.162**	**0.381**	**0.079**	**0.412**	**0.659**	**0.606**	**0.175**	**0.575**	**0.809**

Species exhibiting disease, 1996–1998

In addition to large increases in the number of diseased stations, there has also been an equally dramatic increase in the number of species exhibiting disease. Whereas in 1996, of the 41 species encountered during the swim surveys, only 11 species (27%) showed signs of disease, by 1997, this total had more than doubled to 28 (68%) (Table 6), an increase of 155%. By 1998, this number has risen to 36 species (85%), more than triple the number in 1996 for an overall increase of 218% in this 24 month period. As in the tally of stations, the largest increase was between 1996 and 1997. With the list already including 85% of all species encountered in the survey (Table 6), future increases will, by definition have to be smaller.

In 1996, four of the 11 diseased species had diseased individuals occurring in only one station (Table

Table 5. The proportion of EPA Coral Reef Monitoring Stations affected by various coral diseases (WH, white diseases; BB, black band disease; OD, other diseases; and TD, total diseases), 1996–1998 (Table 4; Fig. 4). Means are listed (with upper and lower 95% confidence limits) for N=40 sites. All disease increases between 1996 and 1998 are highly significant at α=0.05 (one-sided test) and power=0.75. With the exception of black band disease, disease increases between 1996 and 1997 are also significant at the 95% confidence level

Year	Disease condition			
	WH	BB	OD	TD
1996	0.04 (0.01–0.08)	0.04 (0.01–0.07)	0.10 (0.05–0.15)	0.16 (0.10–0.21)
1997	0.38 (0.31–0.46)	0.08 (0.03–0.12)	0.41 (0.33–0.48)	0.66 (0.59–0.74)
1998	0.61 (0.53–0.68)	0.18 (0.12–0.23)	0.58 (0.50–0.65)	0.80 (0.74–0.86)

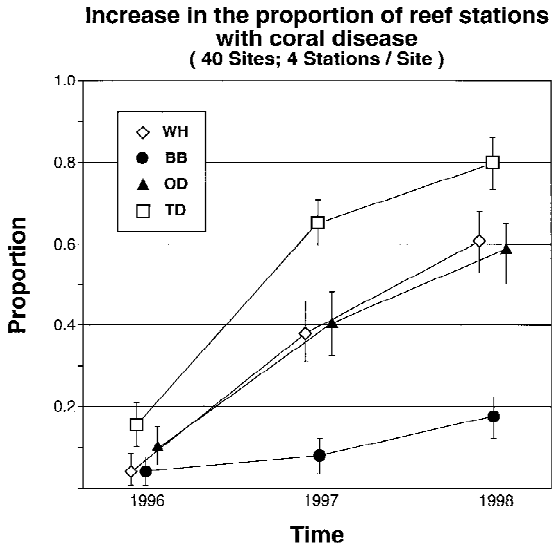

Figure 4. Increase in the proportion of EPA Coral Reef Monitoring stations with coral disease. The mean (±S.D.; N=40 sites) are plotted for black band (BB, closed circles), other diseases (OD, closed triangles), white diseases (WH, open diamonds), and total disease (TD open squares) from data in Table 5.

Table 6. The number of coral species in the EPA Coral Reef Monitoring Stations in the Florida Keys with coral diseases, including white diseases (WH), black band disease (BB), other diseases (OD), and total diseases (TD), and their percent increase, 1996–1998. The percent of affected species is listed in parentheses, based on an S value of 41 species (Table 7)

Year	Disease condition			
	WH	BB	OD	TD
Number of Coral Species in the Florida Keys with Disease				
1996	**3** (7%)	**2** (5%)	**8** (20%)	**11** (27%)
1997	**22** (54%)	**5** (10%)	**22** (54%)	**28** (68%)
1998	**29** (68%)	**7** (17%)	**28** (68%)	**36** (85%)
Increase (1996–1997)	633%	100%	175%	155%
Increase (1997–1998)	27%	75%	27%	25%
Increase (1996–1998)	**833%**	**250%**	**250%**	**218%**

7). The remaining seven species had diseased individuals found in as few as two stations (*Diploria strigosa, Dichocoenia stokesi, Montastraea cavernosa, Siderastrea siderea* and *Stephanocoenia michelinii*) but only at most in six stations (*Montastraea annularis*). By 1997, 10 species had diseased individuals in more than 6 stations, and by 1998, 16 species did so. Furthermore, whereas only two species exhibited signs of more than one category of disease in 1996, 18 did so in 1997, and 22 in 1998. Some species, such as *M. annularis, M. cavernosa* and *S. siderea* had colonies infected with diseases in every disease category in 1997. This never occurred in 1996. By 1998, these latter species plus *Colpophyllia natans*, *Diploria strigosa* and *Stephanocoenia michelinii* had colonies affected by all three disease categories (Table 7). These increases are plotted for several different species for white diseases (Fig. 5a), black band (Fig.

Table 7. Incidence of coral disease by species, and the number of stations exhibiting each of the three disease conditions (WH, white disease; BB, black band disease; and OD, other diseases), 1996–1998 (see Fig. 5)

Species	1996			1997			1998		
	WH	BB	OD	WH	BB	OD	WH	BB	OD
1. *Acropora cervicornis*	1			13		1	18		1
2. *Acropora palmata*	4			19			20		
3. *Agaricia agaricites*				10		1	13		4
4. *Agaricia fragilis*							2		1
5. *Agaricia lamarcki*									
6. *Cladocora arbuscula*							4		
7. *Colpophyllia natans*				5		1	8	6	12
8. *Dendrogyra cylindrus*				1		1	1		1
9. *Dichocoenia stokesi*	2			18		4	31		14
10. *Diploria clivosa*									1
11. *Diploria labyrinthiformis*			1	5		4	5		5
12. *Diploria strigosa*		1	1	1		7	1	1	3
13. *Eusimilia fastigiata*				1			4		6
14. *Favia fragum*				1	1				3
15. *Isophyllastraea rigida*									
16. *Isophyllia sinuosa*									
17. *Leptoseris cucullata*				6		1			3
18. *Madracis decactis*				1		1	3		2
19. *Madracis mirabilis*				1		1	2	1	
20. *Manicina areolata*				1			1		
21. *Meandrina meandrites*						3	3		7
22. *Millepora alcicornis*				1			1		
23. *Millepora complanata*									1
24. *Montastraea annularis*		6	6	10	2	11	20	16	22
25. *Montastraea cavernosa*			2	3	2	1	2	9	30
26. *Mussa angulosa*									3
27. *Mycetophyllia aliciae*						1	1		
28. *Mycetophyllia danaana*							1		6
29. *Mycetophyllia ferox*			1			3	2		3
30. *Mycetophyllia lamarckiana*				1		4	3		10
31. *Oculina diffusa*							8		
32. *Porites astreoides*			3	13		8	7		20
33. *Porites branneri*									
34. *Porites porites*				2		1	1		4
35. *Scolymia cubensis*						1			1
36. *Scolymia lacera*									
37. *Siderastrea radians*						1	2		2
38. *Siderastrea siderea*			2	15	6	36	8	5	42
39. *Solenastrea bournoni*						1	1		
40. *Solenastrea hyades*									3
41. *Stephanocoenia michelinii*			2	2	1		3	2	23

5b) and other diseases (Fig. 5c). These data are striking in their uniformity of change: from 1996 to 1998, all coral species increase in all disease categories in all years. For some of these species, such as *Acropora palmata* (Fig. 5a), diseased individuals are now found in 20 out of 21 stations where this species is present. Together, these data portray a pattern of serious increases in the both the geographic distribution and rate of spread of coral diseases throughout the Florida Keys.

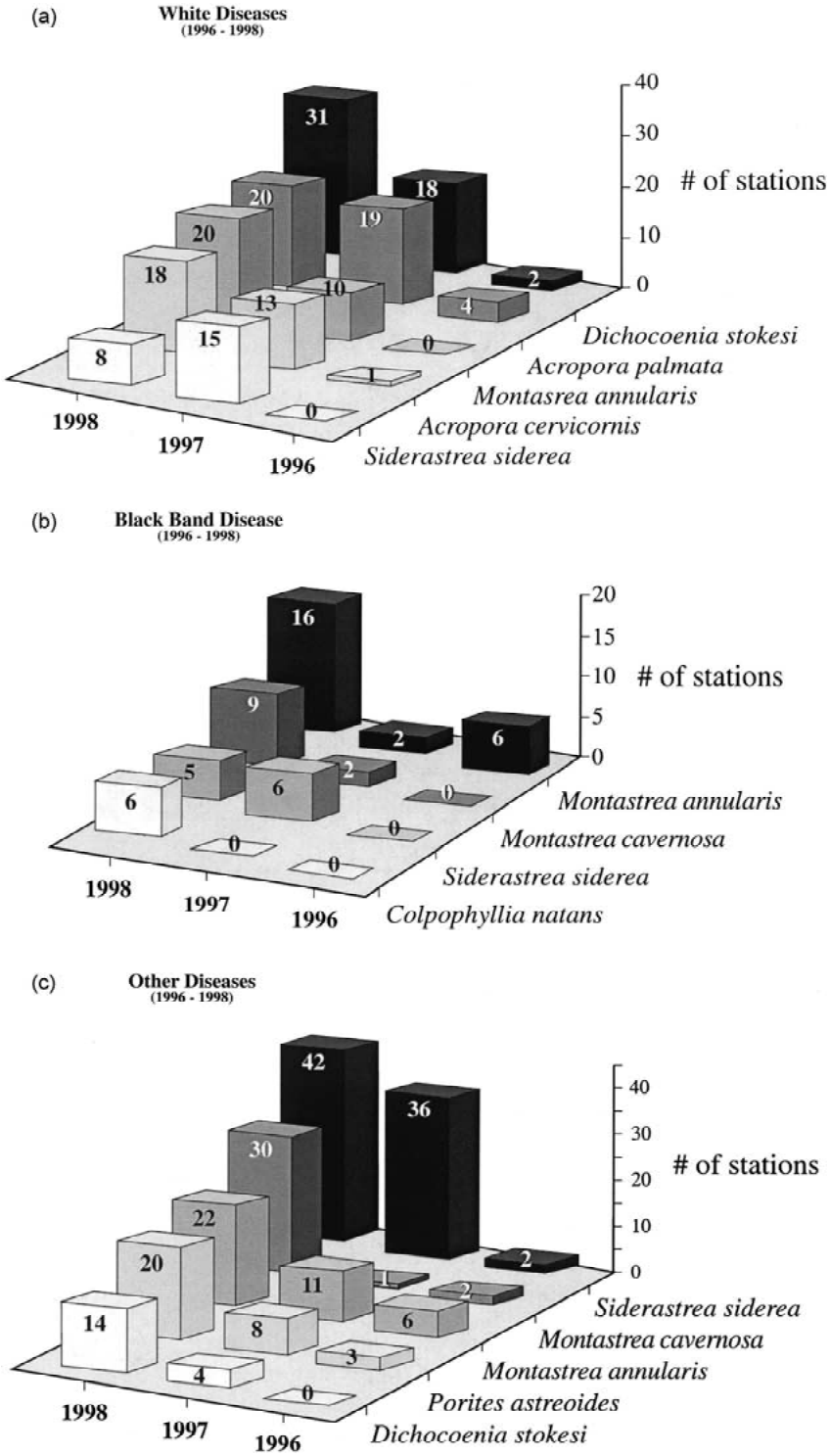

Figure 5. (a–c) Three dimensional plots by year (1996–1997) of the number of coral reef monitoring stations affected by each of the three disease types (White Diseases, Fig. 5a, top; Black Band Disease, Fig. 5b, middle; and Other Diseases, Fig. 5c, bottom) for several coral species in the Florida Keys (Table 7).

Patterns of disease distribution in the Florida Keys, 1996–1998

The EPA Coral Reef Monitoring Project creates a rich data base on the geographic distribution of coral disease in the Florida Keys. A specific example of this can be seen for the spread of white pox on *Acropora palmata* (Figs 3 and 6a). In 1996, four stations had at least one example of white pox on *Acropora palmata*; in 1997, that number had risen by a factor of almost five to 19 stations (Figs 5a and 6a). In most cases where white pox was present in 1996 on one station at a site, by 1997, it had spread to all the stations at that site (Fig. 6a), demonstrating its ability to spread rapidly between as well as within sites. This disease was present throughout the Keys in very low abundance prior to 1996; 1 year later, it has become common everywhere. These data unequivocally demonstrate that between 1996 and 1998, there has been a major outbreak of white pox in the Florida Keys. This fact is also demonstrated in Figure 3, which shows that the prevalence of white pox has increased on *A. palmata* to the point where it now appears in our EPA CRMP survey videos.

A contrasting pattern can be seen in the distribution and spread of black band (Fig. 6b). Like white pox, this condition increased in the Florida Keys (although at a much lower rate, from only seven to 12 stations; Table 3). Unlike white pox, however, most of the increase was between sites rather than within sites (Fig. 6b). Not one of the seven stations with black band in 1996 had it in 1997; the 12 stations with black band in 1997 are entirely new stations. In 1996, the few incidences of black band surveyed were distributed in the Upper Keys (Grecian Rocks), and in the Middle Keys (Looe Key) (Fig. 6b). In 1997, they were distributed from the Upper Keys (Molasses Reef) to the Lower Keys (Sand Key, Rock Key, and the Sambo's). To the extent that there was a disease 'hot spot' in 1996, it was on Looe Key Reef in the Middle Keys where half of the shallow stations exhibited the disease. By 1997, the area of highest black band disease had shifted to the Upper Keys on Conch and Molasses Reefs, where again approximately half of these stations contained the disease.

In six of the seven stations with black band in 1996, this disease was manifested on *Montastraea annularis* (Table 7). One of these seven stations had one example of black band destroying a single colony of *Diploria strigosa*. By 1997, this simple pattern of black band infection on *M. annularis* had changed (Table 7; Fig. 5b). While most stations with the disease again had one species in common, this time the species in common was *Siderastrea siderea*, not *M. annularis* (Table 7; Fig. 5b). In 1996, not a single specimen of *S. siderea* in our station inventories was infected with black band.

The black band data reveal two other patterns. Since 1997, black band has been much less widespread than either white diseases or other diseases in the Florida Keys. Also, its rate of increase has been much slower than the other two disease categories.

Whereas 16 stations showed the presence of other diseases in 1996 (10% of all stations), this number has risen to 65 (41%) in 1997, and 92 (58%) by 1998, an increase of 475% (Table 3). In 1996, only eight coral species were affected by other diseases (Table 6), and OD lesions occurred mostly on either *Montastraea annularis* or *Porites astreoides* (Table 7). By 1997, 22 species were affected, and, by 1998, 28 species were affected, a 250% increase (Table 6). While both of the afore mentioned species are again high on the list of species with affected colonies, *Siderastrea siderea* emerges as the most commonly affected species in both 1997 and 1998 (Table 7; Fig. 5c). Other species, such as *Colpophyllia natans, Dichocoenia stokesi, Mycetophyllia lamarckiana*, *Stephanocoenia michelinii* and *Montastraea cavernosa* which were unaffected in 1996, are now heavily impacted by these diseases (Table 7; Fig. 5c). Other diseases were mostly confined to the Middle and Lower Keys in 1996. By 1998, other diseases are still most prevalent in the Lower Keys, but have spread northward to include the Upper Keys as well (Table 4). One of the striking patterns visible in the disease incidence data reported in Table 7 is that whereas only two species were afflicted by more than one disease in 1996, 22 species were by 1998.

Live coral cover

The EPA coral reef monitoring team has been observing loss due to coral disease on several sites in the Florida Keys since the inception of the project (Figs 2 and 3). Nowhere is this more obvious than on the deep fore-reef site at Carysfort Reef (Figs 7 and 8). Percent live coral cover at this site has plummeted from an average of 13.3% at the beginning of the survey to 5.3%, an overall decrease of 60% in the living coral resources of this reef (Fig. 9). There has been a decrease in living coral cover at all four stations (Fig. 9). Both macroalgae and turf algae have increased (Table 8), with the consequence that space formerly occu-

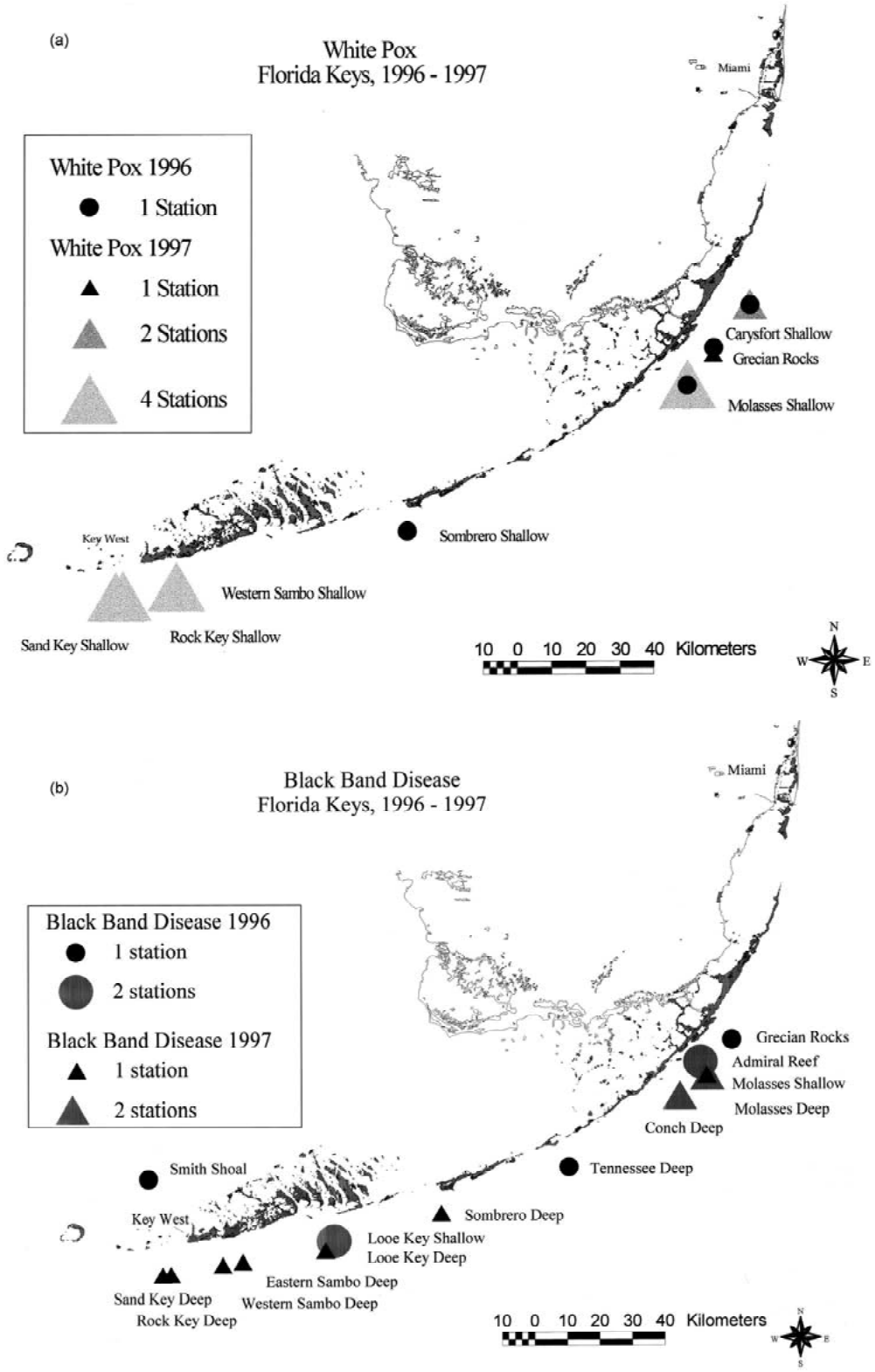

Figure 6. (a, b) The pattern of spread of coral disease between 1996 and 1997 for white pox on *Acropora palmata* (Fig. 6a) and black band (Fig. 6b). Symbols define (a) the location and (b) the number of stations within a site with that disease condition for 1996 or 1997.

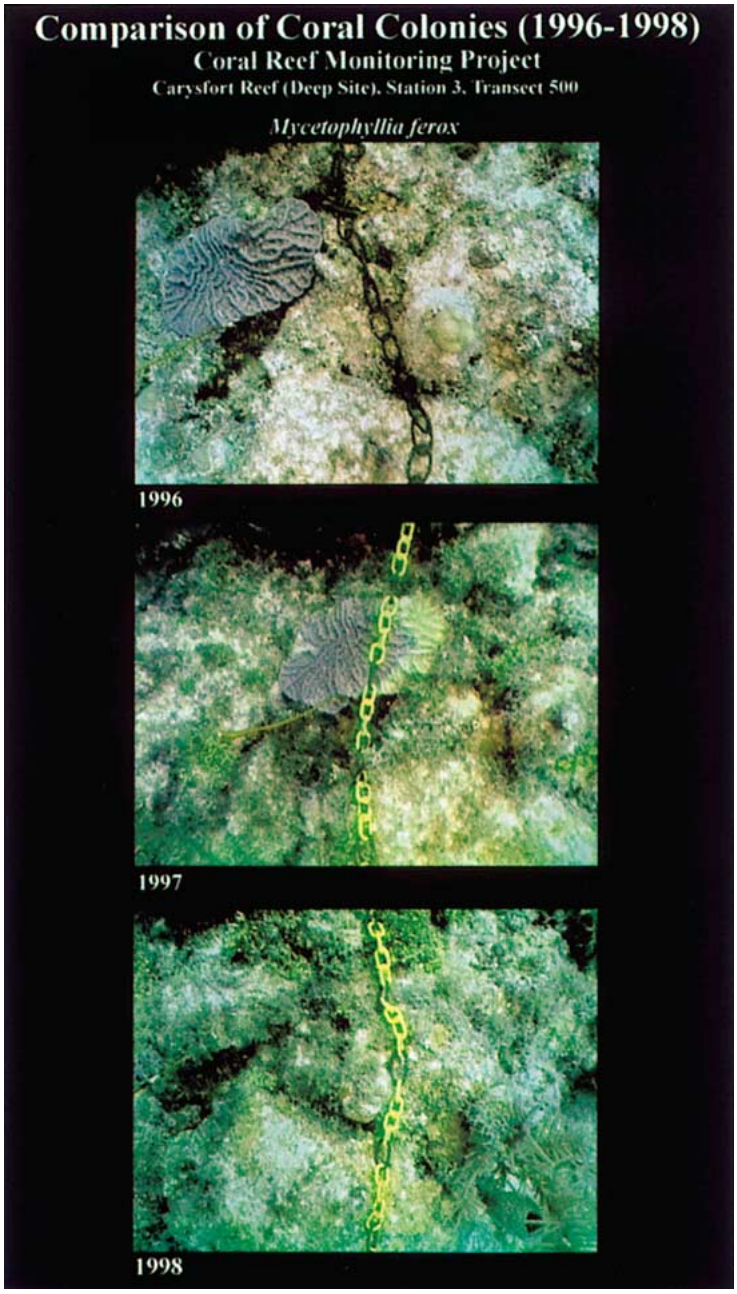

Figure 7. Comparison of frame-grabbed images of corals from the Carysfort Deep Reef monitoring site, 1996–1998. On Station 3, we monitored the loss of *Mycetophyllia ferox* due to white disease from its healthy state in 1996 (top), through its infection in 1997 (middle), and its subsequent death by 1998 (bottom). These visual observations are confirmed by the quantitative information presented in Tables 8c for this species in this station.

pied by coral is now covered with algae (Fig. 7). It is impossible to assess the ecosystem impact of this substantial loss of coral abundance and biodiversity. This rate of loss coral is unsustainable. It is converting one of the most topographically complex and well developed coral reefs in the Florida Keys (Dustan, 1977; Dustan & Halas, 1987) from a coral-dominated reef into an algal-dominated hardbottom.

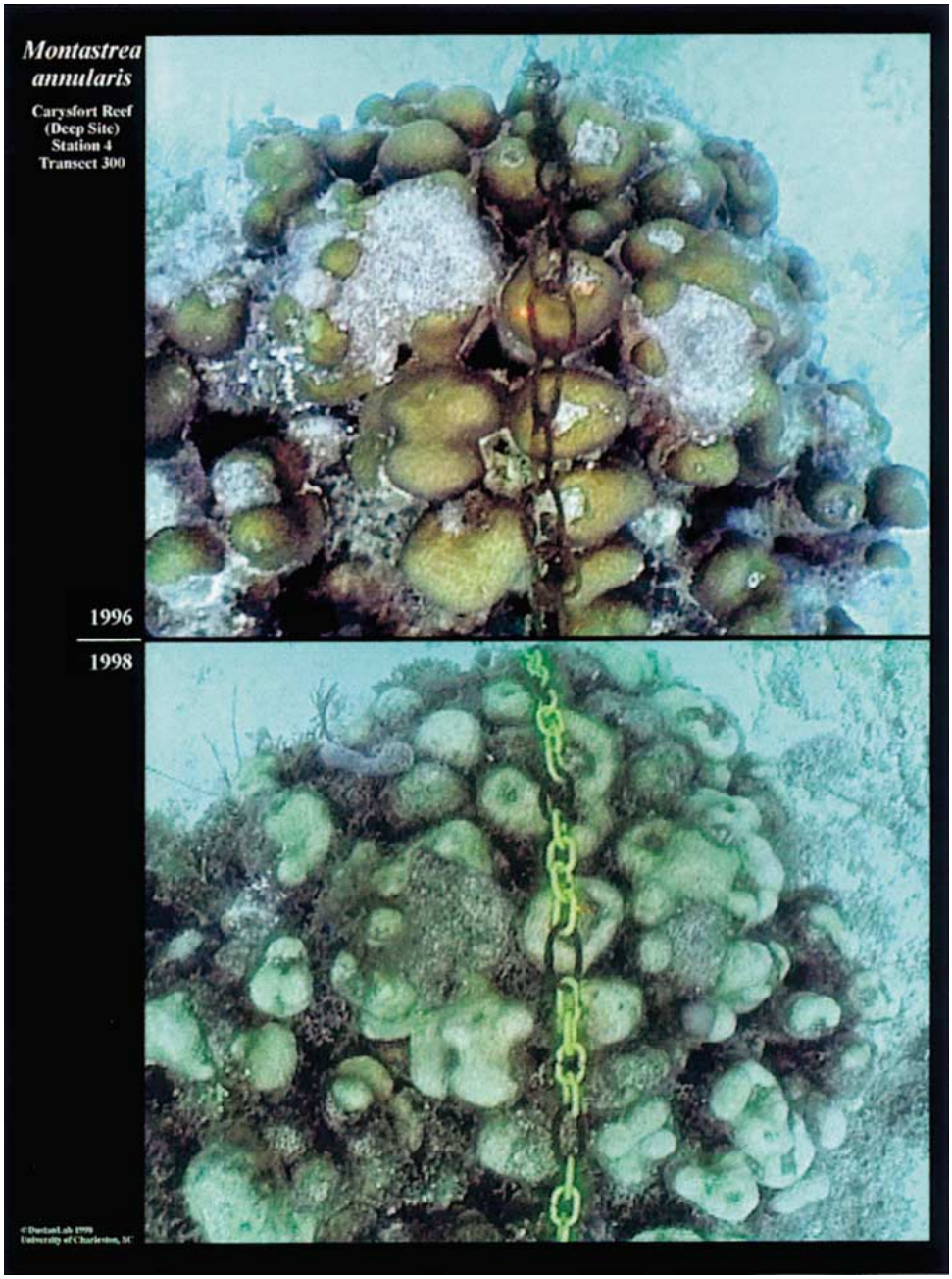

Figure 8. Comparison of frame-grabbed images of corals from the Carysfort Deep Reef monitoring site, 1996–1998. In 1996 (top), this large colony of *Montastraea annularis* from Station 4 became infected with white plague disease. We monitored the loss of its tissue due to disease until the death of the colony in 1998 (bottom). Because coral disease and bleaching can often be confused, coral loss may be attributed to bleaching when it is actually due to disease. This colony of *Montastraea annularis* had already died by early July, 1998. If we had not been monitoring this colony carefully, its death in 1998 could easily have been attributed to the world-wide coral bleaching event which occurred in late summer of that year. These visual observations are confirmed by the quantitative analysis on the reduction in abundance of *Montastraea annularis* presented in Table 8d for this station.

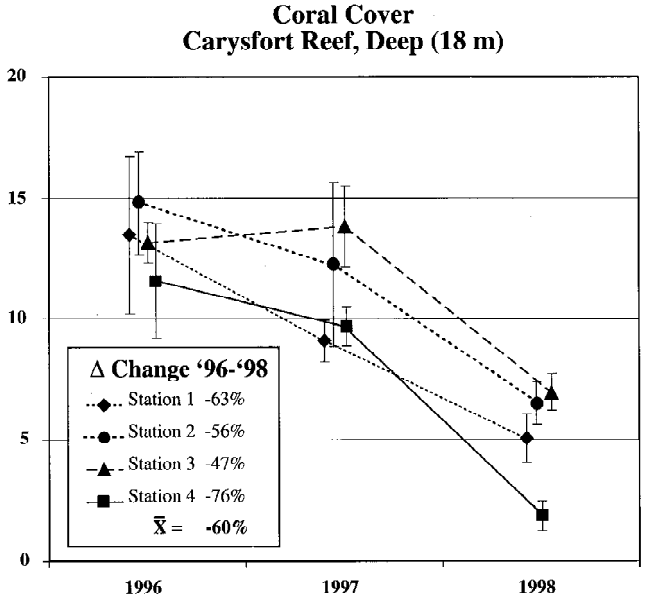

Figure 9. Means and standard errors of percent live cover for at the four Carysfort Deep CRMP Stations, 1996–1998 (Table 8).

Discussion

Increase in the number of stations with disease

The assertion that large-scale marine epidemics are a relatively new phenomena and that the prevalence of disease in marine ecosystems is increasing is based on a survey of proxy data and is controversial (Epstein, 1998; Harvell et al., 1999; Hayes et al., 2001). The aspect that makes the assertion controversial is a lack of studies with historically relevant data taken in a statistically defensible manner. Because of the initial random selection of our sites, the careful recensus techniques used, and the magnitude of the changes observed (Fig. 4), we can conclude from our study that the trends we observed towards an increase in the distribution of disease are both real and ecosystem-wide for corals in the Florida Keys. Between 1996 and 1998, the number of stations with diseased coral increased by a factor of five, from 26 to 131 stations (Table 3). At present, there are no equivalent data against which to compare our findings.

While modest increases occurred in the distribution of black band disease, major increases occurred in the distribution of both white and other diseases. White diseases increased their distribution by more than an order of magnitude, from seven to 97 stations, and other diseases rose by more than a factor of five, from 16 to 92 stations (Table 3).

Whereas in 1996, black band constituted 22% of all disease observations, seven of 32 disease records (Table 7), by 1998 this had fallen to 9% (40 of 448 disease records). This decrease is not due to black band becoming rarer, but rather due to other diseases becoming commoner. Furthermore, whereas in 1996, each instance of black band was in a separate station [seven stations (Table 3) and seven incidences by species (Table 7)], in 1998, several stations had more than one species infected with black band, which is why 40 individual observations (Table 7) do not equal 40 stations (Table 3).

Patterns of disease spread

In several studies elsewhere in the Caribbean, black band has been shown to be distributed in a non-random, clumped distribution (Rutzler & Santavy, 1983; Antonius, 1985; Edmunds, 1991; Bruckner & Bruckner, 1997a). Our data suggest a more haphazard spread of this disease in the Florida Keys (Fig. 6b), but additional sampling, probably monthly between April and December, would be required to test this idea. At this point in time, black band does not appear to be as great a threat in the Florida Keys as it has been elsewhere in the Caribbean (Groshold & Ruiz, 1997; Bruckner & Bruckner, 1997b) or possible also as it once was in Florida (Kuta & Richardson, 1996).

In contrast to black band, our data for the spread of white pox on *Acropora palmata* matches the pattern

Table 8. (a–d) The percent cover of scleractinian corals and other biota at the CRMP Carysfort Deep Site (18 m) for all four stations, 1996–1998. Mean and standard deviation is calculated from random points counted in each of three 20 m transects layed parallel across each station. The average change in live coral cover at this site is −60% in the 2-year interval from 1996 to 1998 (Fig. 9)

Species	1996		1997		1998	
	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)
Carysfort Reef Deep						
Station 1 (Lat. 25° 13.2481′; Long. 80° 12.5915′)						
Live Coral Cover (1996–1998)						
Macroalgae	27.72	4.89	29.11	4.96	19.76	5.65
Hydrocorallia	0.00	0.00	0.33	0.30	0.12	0.10
Octocorallia	9.16	3.08	10.42	2.17	9.17	4.57
Porifera	4.19	0.60	1.92	0.80	0.82	0.37
Substrate	45.43	2.66	49.12	6.39	65.08	5.22
Acropora cervicornis	0.00	0.00	0.06	0.11	0.00	0.00
Agaricia agaricites	1.25	0.75	0.66	0.65	0.06	0.10
Agaricia lamarcki	0.00	0.00	0.00	0.00	0.39	0.67
Dichocoenia stokesi	0.00	0.00	0.06	0.11	0.00	0.00
Meandrina meandrites	0.19	0.20	0.00	0.00	0.00	0.00
Montastraea annularis	6.54	4.27	3.89	1.40	1.46	0.33
Montastraea cavernosa	0.91	0.71	0.83	0.54	0.42	0.43
Mycetophyllia aliciae	0.39	0.52	0.13	0.23	0.28	0.35
Mycetophyllia danaana	0.06	0.10	0.06	0.11	0.06	0.10
Mycetophyllia ferox	0.11	0.19	0.00	0.00	0.00	0.00
Porites astreoides	0.33	0.33	0.64	0.39	0.41	0.12
Porites porites	0.40	0.70	0.06	0.11	0.06	0.10
Siderastrea siderea	1.90	1.01	1.03	0.79	1.81	0.93
Stephanocoenia michelinii	0.00	0.00	0.00	0.00	0.12	0.21
Scleractinia	1.40	0.88	1.67	0.38	0.00	0.00
Total Scleractinian Cover	**13.50**	**5.63**	**9.10**	**1.47**	**5.06**	**1.75**
Percent Coral Loss 1996–1998	**−63%**					

(b) Percent cover of scleractinian corals and other biota, Station 2, Carysfort Reef

Carysfort Reef Deep						
Station 2 (Lat. 25° 13.2481′; Long. 80° 12.5915′)						
Live Coral Cover (1996-1998)						
Macroalgae	22.13	5.85	32.94	3.29	25.30	2.21
Hydrocorallia	0.00	0.00	0.33	0.44	0.04	0.06
Octocorallia	7.51	2.32	10.11	2.94	8.02	0.98
Porifera	3.73	2.49	2.28	1.27	1.20	0.17
Substrate	51.85	9.54	41.56	2.80	58.92	3.98
Zoanthidea	0.00	0.00	0.56	0.96	0.04	0.06
Agaricia agaricites	0.24	0.11	0.11	0.19	0.18	0.22
Colpophyllia natans	0.61	0.79	0.22	0.38	0.08	0.13
Diploria labyrinthiformis	0.48	0.46	1.50	1.30	0.04	0.06
Diploria strigosa	0.13	0.22	0.00	0.00	0.39	0.42

Continued on p. 18

Table 8. Continued

Species	1996		1997		1998	
	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)
Montastraea annularis	8.04	4.79	6.83	2.60	2.47	0.79
Montastraea cavernosa	1.17	0.72	0.33	0.29	0.59	0.47
Mycetophyllia aliciae	0.17	0.29	0.11	0.19	0.12	0.21
Mycetophyllia danaana	0.13	0.22	0.11	0.10	0.00	0.00
Mycetophyllia ferox	0.00	0.00	0.00	0.00	0.08	0.14
Porites astreoides	0.00	0.00	0.22	0.19	0.31	0.26
Siderastrea radians	0.11	0.19	0.00	0.00	0.00	0.00
Siderastrea siderea	0.49	0.30	1.83	1.30	1.53	0.29
Stephanocoenia michelinii	0.06	0.10	0.00	0.00	0.00	0.00
scleractinia	3.17	0.34	0.94	0.25	0.71	0.13
Total Scleractinian Cover	**14.78**	**3.64**	**12.22**	**5.84**	**6.50**	**1.80**
Percent Loss 1996–1998	**−56%**					

(c) Percent cover of scleractinian corals and other biota, Station 3, Carysfort Reef

Carysfort Reef Deep						
Station 3 (Lat. 25° 13.3599′; Long. 80° 12.5218′)						
Live Coral Cover (1996–1998)						
Macroalgae	9.18	4.92	19.95	3.75	16.17	4.91
Hydrocorallia	0.07	0.12	0.48	0.62	0.18	0.18
Octocorallia	7.44	0.36	6.29	2.52	6.55	0.93
Porifera	1.18	0.47	0.96	0.64	0.86	0.66
Substrate	68.32	3.03	58.48	3.56	69.27	5.92
Zoanthidea	0.63	0.93	0.00	0.00	0.00	0.00
Agaricia agaricites	0.14	0.12	0.24	0.24	0.31	0.11
Colpophyllia natans	1.40	1.01	0.48	0.47	0.00	0.00
Diploria labyrinthiformis	0.00	0.00	0.00	0.00	0.51	0.24
Montastraea annularis	8.38	1.88	9.12	2.42	4.64	1.66
Montastraea cavernosa	0.82	0.81	1.21	0.75	0.83	0.85
Mycetophyllia aliciae	0.07	0.12	0.16	0.14	0.00	0.00
Mycetophyllia danaana	0.00	0.00	0.00	0.00	0.06	0.10
Mycetophyllia ferox	0.21	0.20	0.00	0.00	0.00	0.00
Mycetophyllia lamarckiana	0.07	0.12	0.08	0.14	0.00	0.00
Porites astreoides	0.20	0.35	0.41	0.71	0.12	0.11
Porites porites	0.55	0.53	0.71	1.23	0.00	0.00
Siderastrea siderea	0.98	0.99	1.11	1.52	0.44	0.29
Solastrea bournoni	0.07	0.12	0.32	0.55	0.06	0.11
scleractinia	0.28	0.13	0.00	0.00	0.00	0.00
Total Scleractinian Cover	**13.18**	**1.53**	**13.84**	**2.89**	**6.97**	**1.28**
Percent Loss 1996-1998	**−47%**					

Continued on p. 19

Table 8. Continued

Species	1996		1997		1998	
	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)	Mean (%)	Std. Dev. (%)
(d) Percent cover of scleractinian corals and other biota, Station 4, Carysfort Reef						
Carysfort Reef Deep						
Station 4 (Lat. 25° 13.3599′; Long. 80° 12.5218′)						
Live Coral Cover (1996–1998)						
Macroalgae	10.66	5.51	35.93	4.43	33.27	5.88
Hydrocorallia	0.93	0.58	1.61	1.09	0.00	0.00
Octocorallia	8.64	4.44	10.29	1.01	10.78	2.62
Porifera	8.56	6.19	2.34	0.27	0.51	0.51
Substrate	59.60	30.33	40.03	4.20	52.66	8.25
Zoanthidea	0.06	0.09	0.12	0.21	0.00	0.00
Acropora cervicornis	0.06	0.09	0.00	0.00	0.00	0.00
Agaricia agaricites	0.42	0.51	0.19	0.02	0.06	0.10
Colpophyllia natans	0.53	0.46	0.12	0.10	0.40	0.55
Eusmilia fastigiata	0.00	0.00	0.06	0.11	0.00	0.00
Montastraea annularis	8.40	4.71	8.75	1.20	1.64	0.70
Montastraea cavernosa	0.06	0.09	0.00	0.00	0.06	0.10
Mycetophyllia danaana	0.00	0.00	0.00	0.00	0.23	0.26
Mycetophyllia ferox	0.06	0.09	0.00	0.00	0.00	0.00
Mycetophyllia lamarckiana	0.06	0.09	0.12	0.20	0.00	0.00
Porites astreoides	0.75	0.65	0.00	0.00	0.17	0.17
Porites porites	0.94	0.87	0.12	0.20	0.17	0.17
Siderastrea siderea	0.29	0.22	0.00	0.00	0.00	0.00
Solastrea bournoni	0.00	0.00	0.00	0.00	0.06	0.10
Stephanocoenia michelinii	0.00	0.00	0.27	0.47	0.00	0.00
scleractinia	0.00	0.00	0.06	0.10	0.00	0.00
Total Scleractinian Cover	**11.56**	**4.09**	**9.62**	**1.41**	**2.78**	**1.03**
Percent Loss 1996–1998	**–76%**					

of spread in another white disease, white plague, on *Dichocoenia stokesi* (Fig. 2). White plague is strongly correlated with coral colony density (Richardson et al., 1998a,b). Several new diseases, including one on the zooanthid coral *Palythoa caribaeorum* (Acosta, 2001), also exhibit this pattern. These data suggest that the white diseases are highly infectious.

Loss of coral biodiversity

The number of coral species exhibiting disease has increased dramatically since 1996 (Table 6). Although our survey protocol does not follow individual coral colonies, the videotaped visual record provides overwhelming support for the assertion that disease is causing the loss of several species within some of our stations in the Florida Keys (Fig. 7). Species within the genus *Mycetophyllia*, including *aliciae*, *danaana* and *ferox* have disappeared from many stations throughout the Florida Keys, including those at the deep Carysfort Reef site. *Acropora cervicornis* has disappeared from six of the 40 stations where it was found in 1996.

Loss of coral cover

The data in Table 8 leave no doubt that coral cover is declining on these reefs. On Carysfort Reef, with the minor exception of *Siderastrea siderea*, every coral

species with an absolute abundance of one-half of one percent (0.5%) or greater experienced severe reductions in their absolute abundance between 1996 and 1998. Every species of *Mycetophyllia* disappeared in at least one station, and staghorn coral, *Acropora cervicornis*, disappeared from all stations throughout the entire site. The video image analyses (Table 8 and Fig. 9) and the diver species inventories (Table 7 and Fig. 4) are very different records, taken by different people using different data-gathering techniques (remote sensing *versus* direct observation). It is gratifying that these two data sets show the same thing: corals which declined in percent cover were also the same ones scored as diseased by the species counters. The visual record further corroborates this (Figs 7 and 8). These are the small details that make up the severe decline seen in Figure 9.

Community level studies suggest the important role that disease can play in coral growth and survival (Dustan, 1977; Dustan & Halas, 1987). Richardson et al. (1998a,b) document the existence of white plague disease on 17 of 43 coral species in the Florida Keys and show that white plague 'Type II' affected as much as 33% of *Dichocoenia stokesi* colonies at the most heavily impacted sites in the Florida Keys. Edmunds (1991) estimates that approximately 4% of the total colony area of *Diploria strigosa* is lost to a single black band disease event. Acosta (2001) estimates that disease is a major source of partial colony mortality in the zooanthid coral *Palythoa caribaeorum*, accounting for at least 10% of the total partial mortality found in the population annually. All species within the genus *Acropora* have been decimated throughout much of their range due to disease (Gladfelter, 1982; Peters et al., 1983; Porter & Meier, 1992; Aronson & Precht, 1997; Aronson et al., 1998; Ritchie & Smith, 1998; Porter et al., 2002). Our own data show that the major frame building coral, *Montastraea annularis*, has declined from roughly 7% to less than 2% cover on Carysfort Reef (Table 8). Other common important reef builders such as *Montastraea cavernosa* and *Siderastrea siderea* also fared poorly (Table 8). Although *M. annularis* is known to be suffering high mortality in Florida (Dustan, 1999) and elsewhere in the Caribbean (Cervino et al., 2001), much less work has been focused on species like *Siderastrea siderea* and *Stephanocoenia michelinii*, and yet in many ways these species may be in more serious trouble (Fig. 5).

We would have preferred to complement this field study with a full epidemiological analysis of the etiology and spread of each of the 12 disease manifestations that constituted our three disease categories (Tables 1 and 9). The unique strength and importance of our study, however, is that it enumerates the incidence of disease annually on all coral species in the community over a wide geographic area. Our data for Carysfort Reef show that common corals are becoming rarer and that many rare corals are going locally extinct. The future of this coral reef site is in jeopardy.

Table 9. Elements of a complete field survey of coral disease

1.	Identification of the pathogen
2.	Geographic distribution of the infections
	a. localized vs.
	b. pandemic
3.	Taxonomic distribution of infections
4.	% of population affected
5.	% of affected individuals which exhibit
	a. partial mortality
	b. whole mortality
	c. genet mortality
6.	Document long-term effects on coral community structure
7.	Location of the pathogen reservoir

Stress and coral disease

Stress lowers resistance to disease. It is known, for instance, that the pathogenicity of *Aspergillus sydowii* on Floridian sea fans is dependent on the status of the host's immune system (Alker et al., 2001). These authors point out that human aspergillosis caused by *A. fumigatus* is often fatal to immuno-compromised individuals (Dixon & Walsh, 1992), but that it is much less frequently so among healthy individuals (Ponton et al., 1991).

One of the central tenets of epidemiology is that temporal and spatial characteristics of disease can provide keys to interpreting causality (Hayes et al., 2001), and the literature is full of references to the suggestion that poor water quality undermines the health of corals (Pastorok & Bilyard, 1985; Peters, 1997; Acosta, 2001). Our data are suggestive, but not definitive on the issue of causality for the recorded outbreak of coral disease in the Florida Keys. In 1996 and 1997, stations in the Upper Keys and the Lower Keys, areas closest to the city centers of Key West and Key Largo, had a higher percentage of stations affected by disease (Table 4) than more distant locations. This spatial

correlation suggests that proximity to human population centers may increase the likelihood of infection. This correlation echos the findings of Kim & Harvell (2002) who state that, "although the probability of infection (prevalence) did not vary by site, if infected, the impact of the disease (virulence) was greater among sea fans near Key West. One possibility is that poor water quality at these sites, as indicated by higher N and P concentrations, and lower water clarity exacerbate disease virulence."

Regardless of poor water quality, by 1998 no areas in the Florida Keys were without infection (Table 4). It is unclear if the initial correlation was spurious or whether water quality deterioration had occurred throughout the Keys to the point where only the remote places, such as the Dry Tortugas (Porter et al., 1999; Santavy et al., 2001) had lower disease prevalence.

Coral disease and the potential affects of global warming

Elevated temperature is a stress. This is clearly manifested during bleaching which is a thermally induced breakdown of host-zooxanthellae symbiosis (Brown, 1997). In general, temperature stress favors disease (Selye, 1955), and corals are no exception to this pattern (Toren et al., 1998). Evidence for this comes from both experimental and correlative studies. Elevated temperature has been shown to accelerate the growth rate and disease activity pathogens (Alker et al., 2001). For instance, *Phormidium corallyticum*, the causative agent of black band disease, is temperature dependent with an optimum of 28–30 °C (Rutzler & Santavy, 1983; Carlton & Richardson, 1995), and *Vibrio* AK-1, which induces bleaching in the coral *Oculina patagonica* (Kushmaro et al., 1996, 1997) also grows faster at elevated temperatures (Kushmaro et al., 1998). Elevated temperature could result in intensified disease activity either by an acceleration of the growth rate of the pathogen, or by a diminution in the efficacy of host defenses. These are not mutually exclusive hypotheses, and both could be operating at the same time. For instance (Alker et al., 2001) demonstrate a significant reduction in the potency of *Gorgonia ventalina* crude extracts against *A. sydowii* infection when assayed at 30 ° *versus* 25 °C. This reduction at the higher temperature may be due to the inactivation of the antifungal compounds. The growth rate of the pathogen (*A. sydowii*), however, was not suppressed at these higher temperatures.

In several cases, bleaching events have been followed by increased outbreaks of disease (Williams & Bunkley-Williams, 1990; Acosta, 2001; see also Fig. 10). The El Nino related bleaching and epizootic among *Briareum asbestinum* fits this model well in that the soft coral mortality (65% of all colonies), occurred immediately following the 1998–1999 bleaching event (Harvell et al., 2001). Although there is little understanding of how bleaching and mortality are causally linked, we would propose that this causal linkage is due to elevated incidence of disease via opportunistic infections.

All current models of global climate change predict a significant increase in sea surface temperature (Kleypas et al., 1999). Given that temperature requirements of most microbial agents are higher than those of their hosts, Alker et al. (2001) predict that increased water temperature will shift host-pathogen interactions in favor of increased pathogenicity of aspergillosis in sea fans. As global warming proceeds, corals will bleach more often, and for longer periods of time (Hoegh-Guldberg, 1999). Many coral diseases are known to be more active during the warmer months of the summer season (e.g. Antonius, 1981; Rutzler & Santavy, 1983; Feingold, 1988; Kuta & Richardson, 1996) and the question remains what result global warming will have if the 'disease season' lengthens. If we connect this physical oceanographic scenario of global warming with its most likely biological consequence, it is easy to predict that coral disease will become even more common and more widespread over the next quarter century. It is highly likely that the increasing disease trends identified in this study will continue, or even accelerate.

Potential ecosystem effects

Changes in the population size, growth and reproduction of a community's primary producers and major framework builders will have far reaching impacts on the community. These changes are especially relevant given the longevous age structure of corals and, as compared to macroalgae, their relatively slow coral recruitment, especially in the Florida Keys (Tougas & Porter, 2002). Edmunds (1991) noted that areas of corals killed by black band disease did not show any scleractinian recruitment after 2 years. Our study demonstrates that in the last years of the 20th century, disease is an important factor changing the composition, structure and probably function of the Florida Keys coral reef ecosystem. Species-level studies else-

where in the Caribbean suggest that this generalization may be true for other coral reef ecosystems as well (Gladfelter, 1982; Edmunds, 1991; Kuta & Richardson, 1996; Aronson & Precht, 1997; Aronson et al., 1998).

Future disease investigations

The Coral Reef Monitoring Project is acutely aware of the desirability of being able to identify all diseases in situ, and therefore supports the laboratory investigations required to satisfy Koch's postulates (Rand, 1995; Table 9). From a field-work perspective, we must assess coral mortality by following the fates of individual colonies through time. Only with this information can the true impact of these newly emergent diseases be quantified. Future research also needs to determine the environmental conditions which result in the onset and spread of diseases, and establish the causal relationship between stress and disease.

The growing awareness of the importance of rapid climate change and the impact this will have on the health of the oceans (Epstein et al., 1998; Harvell et al., 1999) was well illustrated by the catastrophic die off of corals following the 1997–1998 El Nino event (Wilkinson et al., 1999) in the Indian Ocean. Alker et al. (2001) point out that, "In light of the possibility that the death of some of these corals resulted from subsequent infections (Harvell et al., 1999), a clear understanding of the factors mediating host pathogen interactions will be essential for better predicting the impacts of global climate change on corals and coral reefs, and better devising appropriate management protocols."

Conclusions

Our data definitively refute the null hypothesis that an increase in coral disease in the Florida Keys is only an *apparent* increase due to increasing observational frequency and awareness. Instead, these data prove that coral disease is becoming much more widespread than in the recent past. Whether reported in terms of (1) the number of stations affected, (2) the number of species affected, or (3) the number of different diseases recorded, corals in the Florida Keys are enduring a plethora of infections. Whether these diseases are the expression of an episodic epizootic with only short term ramifications, or a pandemic with long-term ramifications is unknown. Further research and monitoring is absolutely critical to discern which of these alternative ecosystem-wide processes is at work. Detailed knowledge of disease distribution may provide clues as to conditions which promote disease infection and spread, and to answer the fundamental question of whether anthropogenic influences are playing a part in this current outbreak of so many diseases on coral reefs in Florida.

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References

Acosta, A., 2001. Disease in Zooanthids: dynamics in space and time. Hydrobiologia 460: 113–130.

Alker, A. P., G. W. Smith & K. Kim, 2001. Characterization of *Aspergillus sydowii* (Thom et Church), a fungal pathogen of Caribbean sea fan corals. Hydrobiologia 460: 105–111.

Antonius, A., 1981. The 'band' diseases in coral reefs. Proc. Third Internat. Coral Reef Symp. 2: 7–14.

Antonius, A., 1985. Black band disease infection experiments on hexacorals and octocorals. Proc. Fifth Internat. Coral Reef Symp. 6: 155–160.

Aronson, R. B. & W. F. Precht, 1997. Stasis, biological disturbance, and community structure of a Holocene coral reef. Paleobiology 23: 326–346.

Aronson, R. B., W. F. Precht & I. G. Macintyre, 1998. Extrinsic control of species replacement on a Holocene reef in Belize: the role of coral disease. Coral Reefs 17: 223–230.

Aronson, R. B. & W. F. Precht, 2000. Herbivory and algal dynamics on the coral reef at Discovery Bay, Jamaica. Limnol. Oceanogr. 45: 251–255.

Berger, L., R. Speare, P. Daszak, D. E. Green, A. Cunningham, C. L. Goggin, R. Slocombe, M. A. Ragan, A. D. Hyatt, K. R. McDonald, H. B. Hines, K. R. Lips, G. Marantelli & H. Parkes, 1998. Chytridiomycosis causes amphibian mortality associated with population declines in the rain forests of Australia and Central America. Proc. Natl. Acad. Sci. U.S.A. 95: 9031–9036.

Brown, B. E., 1997. Coral bleaching: Causes and consequences. Coral Reefs 16: 129–138.

Bruckner, A. W. & R. J. Bruckner, 1997a. The persistence of black band disease in Jamaica: impact on community structure. Proc. Eighth Internat. Coral Reef Symp. 1: 601–606.

Bruckner, A. W. & R. J. Bruckner, 1997b. Outbreak of coral disease in Puerto Rico. Coral Reefs 16: 260.

Carlton, R. G. & L. L. Richardson, 1995. Oxygen and sulfide dynamics in a horizontally migrating cyanobacterial mat: black band disease of corals. FEMS Microbiol. Ecol. 18: 155–162.

Cervino, J. M., T. J. Goreau, I. Nagelkerken, G. W. Smith & R. Hayes, 2001. Yellow band and dark spot syndromes in Caribbean corals: distribution, rate of spread, cytology, and effects on abundance and division rate of zooxanthellae. Hydrobiologia 460: 53–64.

Clarke, K. R., 1993. Non-parametric multivariate analyses of change in community structure. Aust. J. Ecol. 18: 117–143.

Dixon, D. M. & T. J. Walsh, 1992. Human pathogenesis. In Bennett, J. & M. Klich (eds), Aspergillus: Biology and Industrial Applications. Butterworth-Heinemann, Boston, MA: 249–267.

Dustan, P., 1977. Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Envir. Geol. 2: 51–58.

Dustan, P., 1999. Coral reefs under stress: Sources of mortality in the Florida Keys. Nat. Res. For. 23: 147–155.

Dustan, P. & J. Halas, 1987. Changes in the reef-coral population of Carysfort Reef, Key Largo, Florida, 1975–1982. Coral Reefs 6: 91–106.

Edmunds, P. J., 1991. Extent and effect of black-band disease on a Caribbean reef. Coral Reefs 10: 161–165.

Epstein, P. R., 1998. Marine ecosystems: Emerging diseases as indicators of change. Harvard Medical School, Boston, MA: 85 pp.

Feingold, J. S., 1988. Ecological studies of a cyanobacterial infection of the Caribbean sea plume *Pseudopterogorgia acerosa* (Coelenterata: Octocorallia). Proc. Fifth Internat. Coral Reef Symp. 3: 157–162.

Gladfelter, W. B., 1982. White band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull. mar. Sci. 32: 639–643.

Goreau, T. J., J. M. Cervino, M. Goreau, R. L. Hayes, L. L. Richardson, G. W. Smith, K. DeMeyer, I. Nagelkerken, J. Garzon-Ferra, D. Gil, G. Garrison, E. Williams, L. Bunkley-Willaims, C. Quirolo, K. L. Patterson, J. W. Porter & K. G. Porter, 1998. Rapid spread of diseases in Caribbean coral reefs. Rev. Biol. Trop. 46: 157–172.

Greenstein, B. J., H. A. Curran & J. M. Pandolfi, 1998. Shifting ecological baselines and the demise of *Acropora cervicornis* in the western Atlantic and Caribbean Province: a Pleistocene perspective. Coral Reefs 17: 249–261.

Groshold, E. D. & G. M. Ruiz, 1997. Evidence for regional adaptation of black band disease at Carrie Bow Cay, Belize. Proc. Eighth Internat. Coral Reef Symp. 1: 579–582.

Hankinson, J. H. & E. J. Conklin, 1996. Water quality protection program for the Florida Keys National Marine Sanctuary: First biennial report to Congress. U.S. EPA Office of Water. Washington, DC: 33 pp.

Harvell, D., K. Kim, C. Quirolo, J. Weir & G. Smith, 2001. Coral bleaching and disease: contributions to 1998 mass mortality in *Briareum asbestinum* (Octocorallia, Gorgonacea). Hydrobiologia 460: 97–104.

Harvell, C. D., K. Kim, Harvell et al. J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, E. K. Lipp, A. D. M. E. Osterhaus, R. M. Overstreet, J. W. Porter, G. W. Smith & G. R. Vasta, 1999. Emerging marine diseases – climate links and anthropogenic factors. Science 285: 1505–1510.

Hayes, M. L., J. Bonaventura, T. P. Mitchell, J. M. Prospero, E. A. Shinn, F. Van Dolah & R. T. Barber, 2001. How are climate and marine biological diseases functionally linked? Hydrobiologia 460: 213–220.

Hayes, R. L. & T. J. Goreau, 1998. The significance of emerging diseases in the tropical coral reef ecosystem. Rev. Biol. Trop. 46: 173–185.

Hoegh-Guldberg, O., 1999. Climate change, coral bleaching, and the future of the world's coral reefs. Mar. Freshwat. Res. 50: 839–866.

Holden, C., 1996. Coral disease hot spot in the Florida Keys. Science 274: 2017.

Hughes, T. P., 1994. Catastrophes, phase shifts, and large scale degradation of a Caribbean coral reef. Science 265: 1547–1551.

Hughes, T. P., A. M. Szmant, R. Steneck, R. Carpenter & S. Miller, 1999. Algal blooms on coral reefs: What are the causes? Limnol. Oceanogr. 44: 1583–1586.

Kim, K. & C. D. Harvell, 2002. Aspergillosis of sea fan corals: Disease dynamics in the Florida Keys. In Porter, J. W. & K. G. Porter (eds), The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL: 813–824.

Kleypas, J. A., R. W. Buddemeier, D. Archer, J. P. Gattuso, C. Langdon & B. N. Opdyke, 1999. Geochemical consequences of increased atmospheric carbon dioxide on coral reefs. Science 284: 118–120.

Kushmaro, A., Y. Loya, M. Fine & E. Rosenberg, 1996. Bacterial infection and coral bleaching. Nature 380: 396.

Kushmaro, A., Y. Loya, M. Fine & E. Rosenberg, 1997. Bleaching of the coral *Oculina patagonica* by *Vibrio* AK-1. Mar. Ecol. Prog. Ser. 147: 159–165.

Kushmaro, A., E. Rosenberg, M. Fine, Y. Ben Haim & Y. Loya, 1998. Effects of temperature on bleaching of the coral *Oculina patagonica* by *Vibrio* AK-1. Mar. Ecol. Prog. Ser. 171: 131–137.

Kuta, K. G. & L. L. Richardson, 1996. Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs 15: 219–223.

Lapointe, B. E., 1997. Nutrient thresholds for bottom up control of macroalgal blooms on coral reefs in Jamaica and southeast Florida. Limnol. Oceanogr. 42: 1119–1131.

Lapointe, B. E., 1999. Simultaneous top-down and bottom-up forces control macroalgal blooms on coral reefs (Reply to the comment by Hughes et al.). Limnol. Oceanogr. 44: 1586–1592.

Lessios, H. A., D. R. Robinson & J. D. Cubit, 1984. Spread of *Diadema* mass mortality through the Caribbean. Science 226: 335–337.

McCallum, H. & A. Dobson, 1995. Detecting disease and parasite threats to endangered species and ecosystems. Trends Ecol. Evol. 10: 190.

McClanahan, T. R. & N. A. Muthiga, 1998. An ecological shift in a remote coral atoll of Belize over 25 years. Environ. Conserv. 25: 122–130.

Nagelkerken, I., K. Buchan, G. W. Smith, K. Bonair, P. Bush, J. Garzon-Ferreira, L. Botero, P. Gayle, C. D. Harvell, C. Heberer, K. Kim, C. Petrovic, L. Pors & P. Yoshioka, 1997a. Widespread disease in Caribbean sea fans: I. Spreading and general characteristic. Proc. Eighth Internat. Coral Reef Symp. 1: 679–682.

Nagelkerken, I., K. Buchan, G. W. Smith, K. Bonair, P. Bush, J. Garzon-Ferreira, L. Botero, P. Gayle, C. D. Harvell, C. Heberer, K. Kim, C. Petrovic, L. Pors & P. Yoshioka, 1997b. Widespread disease in Caribbean sea fans: II. Patterns of infection and tissue loss. Mar. Ecol. Prog. Ser. 160: 255–263.

Overton, W. S, D. White & D. L. Stevens, 1991. Design report for the Environmental Monitoring Assessment Program (EMAP). EPA/600/3-91/053 US EPA, Washington, DC: 85 pp.

Pastorok, R. & G. Bilyard, 1985. Effects of sewage pollution on coral-reef communities. Mar. Ecol. Prog. Ser. 21: 175–189.

Peters, E. C., 1997. Diseases of coral reef organisms. In Birkeland, C. (ed.), Life and Death of Coral Reefs. Chapman and Hall, New York, NY: 114–139.

Peters, E. C., P. P. Yevich & J. J. Oprandy, 1983. Possible causal agent of 'white band disease' in Caribbean acroporid corals. J. Invertebr. Pathol. 41: 394–396.

Ponton, J., G. Quindos & R. Cisterna, 1991. Humoral responses of the host to fungi. In Arora, D. K., L. Ajello & K. Murkerj (eds), Handbook of Applied Mycology, Vol. 2 Humans, Animals, and Insects. Marcel Dekker, Inc., New York, NY: 289–340.

Porter, J. W., V. Kosmynin, K. L. Patterson, K. G. Porter, W. C. Jaap, J. L. Wheaton, K. Hackett, M. Lybolt, C. P. Tsokos, G. Yanev, D. M. Marcinek, J. Dotten, D. Eaken, M. Patterson, O. W. Meier, M. Brill & P. Dustan, 2002. Detection of coral reef change by the Florida Keys Coral Reef Monitoring Project. In Porter, J. W. & K. G. Porter (eds), The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL: 749–769.

Porter, J. W., S. K Lewis & K. G. Porter, 1999. The effects of multiple stressors on the Florida Keys coral reef ecosystem: a landscape hypothesis and a physiological test. Limnol. Oceanogr. 44: 941–949.

Porter, J. W. & O. W. Meier, 1992. Quantification of loss and change in Floridian reef coral populations. Am. Zool. 32: 625–640.

Porter, J. W. & J. I. Tougas, 2001. Reef ecosystems: Threats to their biodiversity. Encyclopedia of Biodiversity 5: 73–95.

Rand, G. M., 1995. Fundamentals of Aquatic Toxicology: Effects, Environmental Fate, and Risk Assessment. Taylor and Francis, Washington, DC: 440 pp.

Richardson, L. L., 1998. Coral diseases: What is really known? Trends Ecol. Evol. 13: 438–443.

Richardson, L. L., R. B. Aronson, W. M. Goldberg, G. W. Smith, K. B. Ritchie, J. C. Halas, J. S. Feingold & S. L. Miller, 1998a. Florida's mystery coral-killer identified. Nature 392: 557–558.

Richardson, L. L., W. M. Goldberg, R. G. Carlton & J. C. Halas, 1998b. Coral disease outbreak in the Florida Keys: Plague Type II. Rev. Biol. Trop. 46: 187–198.

Ritchie, K. B. & W. G. Smith, 1998. Description of type II white band disease in acroporid corals. Rev. Biol. Trop. 46: 199–203.

Roblee, M. B., T. R. Barber, P. R. Carlson, M. J. Durako, J. W. Fourquerean, L. K. Muehlstein, D. Porter, L. A. Yarbro, R. T. Zieman & J. C. Zieman, 1991. Mass mortality of the tropical seagrass *Thalassia testudinum* in Florida Bay. Mar. Ecol. Prog. Ser. 71: 297–299.

Rützler, K. & D. Santavy, 1983. The black band disease of Atlantic reef corals. I. Description of the cyanophyte pathogen. Pubb. Staz. Zool. Napoli Mar. Ecol. 4: 301–319.

Santavy, D. L., E. Mueller, E. C. Peters, L. MacLaughlin, J. W. Porter, K. L. Patterson & J. Campbell, 2001. Quantitative assessment of coral diseases in the Florida Keys: strategy and methodology. Hydrobiologia 460: 39–52.

Santavy, D. L., E. C. Peters, C. Quirolo, J. W. Porter & N. Bianchi, 1999. Yellow-blotch disease outbreak on reefs of the San Blas Islands, Panama. Coral Reefs 18: 97.

Selye, H., 1955. Stress and disease. Science 122: 625–631.

Toren, A., L. Landau, A. Kusmaro, Y. Loya & E. Rosenberg, 1998. Effects of temperature on adhesion of *Vibrio* strain AK-1 to *Oculina patagonica* and on coral bleaching. Appl. Envir. Microbiol. 64: 1379–1384.

Tougas, J. I. & J. W. Porter, 2002. Differential coral recruitment patterns in the Florida Keys. In Porter, J. W. & K. G. Porter (eds), The Everglades, Florida Bay, and Coral Reefs of the Florida Keys. CRC Press, Boca Raton, FL: 789–811.

Warwick, R. M. & K. R. Clarke, 1991. A comparison of some methods for analyzing changes in benthic community structure. J. mar. biol. Acc. U.K. 71: 225–244.

Wilkinson, C., O. Linden, H. Cesar, G. Hodgson, J. Rubens & A. E. Strong, 1999. Ecological and socioeconomic impacts of 1998 coral mortality in the Indian Ocean: an ENSO impact and a warning of future change? Ambio 28: 188–196.

Williams, E. & L. Bunkley-Williams, 1990. The world-wide coral reef bleaching cycle and related sources of coral mortality. Atoll Res. Bull. 335: 1–63.